Remarks/Arguments

The amendment to the specification is a formal nature, correcting an obvious typographical error.

Prior to entry of the foregoing amendment, claims 1, 4-14, and 23 were pending in this application, claims 8, 9, and 11-13 having been withdrawn from consideration. Claims 1 and 23 have been amended, and claims 8-14 have been canceled. The amendments of claims 1 and 23 are fully supported by the specification as originally filed, for example at Figure 2, and page 68, lines 28-29 of the specification, and do not introduce new matter.

All amendments were made without prejudice. Applicants specifically reserve the right to pursue any subject matter currently canceled in one or more continuing applications.

Restriction Requirement

Applicants note the finality of the restriction requirement communicated in the previous Office Action, modified by the rejoinder of SEQ ID NO: 8 in Group 14. Applicants further confirm the oral election of the invention of Group 1 identified on page 4 of the present Office Action.

Priority

Applicants disagree with the Examiner's finding that the present claims would be entitled only to the priority of August 5, 1994 of application Serial No. 08/286,846. All subject matter claimed in the present application is fully supported by the disclosure of application Serial No. 08/215,139 (the '139 application) filed on March 18, 1994. Specifically, the trkC sequence of SEQ ID NO: 6 is shown in Figure 2 of the '139 application. Aberrant neuronal sprouting is discussed in the passage bridging pages 87 and 88 of the '139 specification. Accordingly, the priority date of the present application is March 18, 1994.

Objection

The specification was objected to because of a typographical error at page 3, lines 5-6. This error has been corrected by the foregoing amendment to the specification.

Rejections

(1) Claims 1, 4-7, 10, 14 and 23 were rejected under 35 USC 112, first paragraph for alleged lack of enablement. Claims 10 and 14 have been canceled. The rejection of the remaining claims is respectfully traversed.

In support of the rejection, the Examiner refers to (1) the existence and different properties of various splice variants of trkC; and (2) the alleged lack of nexus between trk C and any disease, in particular aberrant neuron sprouting. Although the Examiner acknowledges the specification's teaching that antagonists of trkC are believed to be useful for treating aberrant neuron sprouting in epilepsy, she states that "there is no explanation as to why this statement is made," and points at the alleged lack of teaching as to which of the various forms of trkC receptor is involved in this process. The Examiner dismisses the teaching of Kim et al. that trkC was strongly expressed in the brain of patients with cerebral cortical dysplasia, one of the important causes of intractable epilepsies, and their suggestion that trkC may have a critical pathogenic role in epileptogenicity in dysplastic neurons of CD, as lacking a "firm prediction as to the involvement of trkC." Finally, the Examiner refers to variables such as stability, half-life, clearance, etc. in support of the unpredictability of successful therapy with *in vivo* administration of the anti-trkC antibodies can be achieved.

Since the claims are now directed to the use of antibodies specifically binding the full-length trkC receptor of SEQ ID NO: 6, without the associated signal sequence, any issues concerning the potentially different biological activities of the various splice forms no longer apply. As to the lack of explanation why trkC antagonists are proposed for the inhibition of aberrant neuron sprouting in epilepsy, applicants note that such explanation is not legally required to meet the enablement requirement.

Furthermore, it is entirely incorrect for the Examiner to dismiss the teaching of the results of Kim et al., just because the authors, in customary style of scientific publications, suggest that trkC may have a critical role in the pathogenesis of epilepsy. It is very rare, if not impossible, to find scientific publications where the authors make entirely definite statements. The important part of the statement referred to is that Kim et al. suggested a "critical" pathogenic role for trkC in epilepsy.

Finally, all assertions concerning the potential issues related to clearance, stability, etc. are believed to be entirely irrelevant for the assessment of enablement. The patent statute does not require that applicants provide details of an invention in a commercially available form. Should this

be a requirement for inventions directed to compounds with therapeutic utility, or to their use, applicants would need to wait with the filing of a patent application until a marketing approval from the FDA is received. Such requirement would be obviously contrary long established case law and the general practice of the Patent Office. Consideration of issues as clearance, half-life, stability, and the like, is within the competence of the FDA, and not the Patent Office.

Applicants submit that based on the teaching of the specification one skilled in the art would have been able to make and use the invention claimed without undue experimentation, therefore, the withdrawal of the present rejection is respectfully requested.

(2) Claims 1, 4-7, 10, 14 and 23 were rejected for alleged lack of written description. Claims 10 and 14 have been cancelled. The rejection of the remaining claims is respectfully traversed.

In support of the rejection, the Examiner relies on the Eli Lilly line of cases and Enzo Biochem, addressing the written description requirement for DNA-related inventions. As the Examiner correctly noted, the present invention does not claim a DNA molecule or its use. Accordingly, the cited cases do not constitute applicable case law. There is nothing in Eli Lilly or Enzo Biochem that would indicate that the CAFC intended the rules established by these cases to be generally applicable, regardless of the nature of the invention. Just the contrary, all cited decisions are replete with statements acknowledging the unique features of DNA molecules. Furthermore, even if the CAFC had implied broader applicability of their holdings, such statements could only have been made *in dictum*, and would not create valid precedent applicable to the present case.

Other than citing unrelated case law, the Examiner has not advanced any evidence or solid scientific arguments why a person of ordinary skill in the art would not have reasonably accepted that applicants were in the possession of the claimed invention at the priority date of this application. Since a *prima facie* showing of lack of adequate written description has not been made, the present rejection should be withdrawn.

(3) Claim 23 has been rejected under 35 USC 112, first paragraph for alleged lack of enablement, since "the specification, while being enabling for a method wherein the condition is associated with elevated NT3, does not reasonably provide enablement for the condition is [sic] associated with elevated endogenous neurotrophin production." Since Claim 23 has been

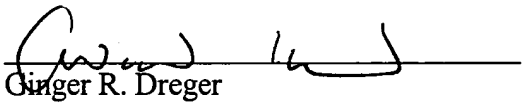
amended to recite conditions associated with elevated NT-3, the withdrawal of the present rejection is respectfully requested.

All claims pending in this application are prima facie condition for allowance, and an early action to that effect is respectfully solicited.

Please charge any fees, including any fees for extension of time, or credit overpayment to Deposit Account No. 08-1641 (**Attorney Docket No.: 39766-0033CPC4C**). Please direct any calls in connection with this application to the undersigned at the number provided below.

Respectfully Submitted,

Date: May 12, 2004



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